

Child goiter

Definition

an enlargement of thyroid gland above the norm for the age (during the ultrasound examination volume of thyroid gland is greater than 2 standard deviations).

Classification

By function

- eufunctional goiter,
- **hypofunctional** goiter
 - bradycardia , weight gain, growth retardation, failure to thrive , dry skin and hair, facial leakage, eyelid swelling, somnolence, fatigue, inefficiency, constipation , premature puberty
- hyperfunctional goiter ,
 - tachycardia , weight loss, failure to thrive, warm, sweaty skin, heat intolerance, exophthalmos, diarrhea

If hyperthyroidism is suspected, the patient must be sent to hospital - there is a risk of heart failure.

By character

1. **diffuse goiter:**
 - lack of iodine (does not occur in the Czech Republic)
 - congenital hormone synthesis disorder (dyshormonogenesis)
 - autoimmune inflammation (Hashimoto's lymphocytic thyroiditis , Graves-Based thyroiditis)
2. **multinodular goiter:**
 - sometimes associated with autoimmune inflammation of thyroid gland
3. **localized node:**
 - thyroid carcinoma (most often differentiated papillary carcinoma or medullary C-cell carcinoma - familial occurrence)
 - 3 / 4 of the solitary nodules are benign - cystic lesion

Diffuse parenchymal goiter

- real thyroid hyperplasia caused by chronic hyperstimulation, especially in chronic iodine deficiency (intake <40 µg / day) and in Basedow's disease .

Struma neonatorum

- etiology : insufficient iodine intake during pregnancy;transplacental transmission of strumigenic substances (PAS, resorcinol); treating the pregnant patient by thyrostatics; transmission of TRAK antibodies from a mother with Basedow's disease;
- intrauterine thyroid hormone deficiency → increased TSH secretion → goiter;
- visible enlargement of the thyroid gland → stridor, breathing difficulties.

Juvenile euthyroid goiter

- iodine deficiency or familial disorder of iodine usage
- development of goiter in puberty, more often in girls;
- eufunctional goiter, homogeneously enlarged → necrosis, cysts, nodules;
- lower amount of thyroid hormones, TSH normal, normal TRH test , thyroid antibodies negative;
- optimization of iodine intake (200 µg / day), if the goiter is refractory - suppression of TSH by thyroxine is indicated.

Disease

Congenital hypothyroidism

- the most common congenital endocrine disease (prevalence 1: 4000);
- thyroid hormones play a key role in brain development, especially by 8 months of age (slightly less so by 3 years of age);
- without substitution treatment, irreversible brain damage occurs - at the clinical diagnosis, the brain is already irreversibly damaged;
- nationwide neonatal screening has been introduced since 1985- determination of TSH level
- etiopathogenesis: **thyroid gland dysgenesis** (agenesis, aplasia, hypoplasia, hemithyroiditis, cystic malformation, ectopy) or **dyshormonogenesis** (disorder of any stage of hormone synthesis or secretion) or rare **isolated congenital central hypothyroidism** (congenital TSH defect - cannot be detected by neonatal

- screening);
- clinical picture without treatment: prolonged neonatal jaundice, failure to thrive, delayed growth rate and bone maturation - late closure of the fontanel, delayed eruption of the lactic dentition, macroglossia, muscle hypotension, omphalocele, constipation, hoarse screaming;
- neonatal goiter or thyroid gland of normal size;
- laboratory findings: \uparrow TSH, \downarrow fT₄; (the central form- \downarrow TSH and fT₄);
- therapy : lifelong L-thyroxine replacement therapy (started as soon as possible).

Autoimmune thyroid gland disease

- the most common acquired thyroid disease in children and adolescents; more often in girls;
- Mostly **lymphocytic (Hashimoto's) thyroiditis**;
- often associated with other autoimmune diseases (type 1 diabetes mellitus, celiac disease) and chromosomal aberrations (Down syndrome, Turner syndrome);
- soft diffuse goiter, USG: diffusely inhomogeneous texture ("pepper and salt"); histology: lymphocytic infiltration of the gland;
- etiopathogenesis : autoantibodies against thyroid peroxidase (anti-TPO) and against human thyroglobulin (anti-hTG);
- clinical picture: the first is the stage of euthyroidism, then the stage of permanent hypothyroidism; there may also be transient hyperthyroidism ("hashitoxicosis");
- without treatment : growth retardation, dyslipidemia, obesity, impaired school achievement, anemia, dry and rough skin, premature pseudopuberty or delayed puberty, myxedema, constipation, bradycardia;
- laboratory findings : \uparrow TSH, \downarrow fT₄;
- therapy : lifelong L-thyroxine replacement therapy.

Neonatal hyperthyroidism (thyrotoxicosis)

- a rare disorder that can be life-threatening for newborns;
- transplacental transmission of maternal antibodies against the TSH receptor in Graves-Basedow-type maternal thyrotoxicosis;
- clinical picture : hyperthyroidism since the fetal period - IUGR, tachycardia, accelerated bone maturation, goiter, exophthalmos, risk of metabolic breakdown and heart failure;
- laboratory finding : \uparrow fT₄ ;
- therapy : antithyroid therapy until maternal antibodies disappear, ie in a descending dose for 2-3 months. [3]

Graves-Basedow thyrotoxicosis

- the most common cause of hyperthyroidism in children; especially in adolescent girls;
- etiopathogenesis : anti-TSH receptor autoantibodies (TRAb, rTSH-ab) that have a thyroid stimulating effect;
- clinical picture : hyperkinetic circulation with tachycardia and systolic hypertension with increased pressure amplitude, weight loss, impaired school achievement, irritability, nervousness, gentle hand tremor, diarrhea, sweating, in 60% orbitopathy with exophthalmos- caused by proliferation of retrobulbar connective tissue by autoimmune stimulation;
- in 75% goiter - strongly perfused, warm, tactile swirl;
- laboratory findings : \downarrow TSH, \uparrow fT₄ ;
- therapy : thyrostatics (methimazole, carbimazole, propithiouracyl), if there are repeated relapses recurrent total thyroidectomy and lifelong L-thyroxine replacement therapy is indicated.

Iodopenic goiter

- our natural diet is low in iodine \rightarrow salt iodization since the 1950s;
- iodine deficiency \rightarrow decreased production of thyroid hormones \rightarrow \uparrow TSH \rightarrow iodopenic goiter;
- endemic cretinism - eradicated.

Examination

Clinical examination

- palpation of the thyroid gland (between the jugular socket and the beginning of the trachea)
- WHO criteria:
 - GRADE 0: the thyroid gland is not palpable
 - GRADE 1: The thyroid gland is palpable but not visible in the normal position of the neck
 - GRADE 2: the thyroid gland is palpable and visible at normal head position

Laboratory examination

- TSH
 - immeasurably low - hyperthyroidism
 - slightly elevated - subclinical hypothyroidism
 - significant increase (tens of mIU / l) - hypothyroidism,
- fT₄
 - significantly increased - hyperthyroidism
 - significantly reduced - hypothyroidism

- antibodies
 - anti-TPO (thyroid peroxidase) - increased anti-TPO indicate autoimmune thyroiditis
 - anti-hTG (human thyroglobulin)
 - TRAK / TRAb (TSH receptor stimulating antibodies) - Graves-Basedow disease

Ultrasound examination of thyroid gland

- we evaluate the size, echotexture of the gland and search for focal changes.

FNAC under ultrasound control

- thin needle aspiration biopsy for subsequent cytological examination

Differential diagnosis of goiter in children

- medial and lateral cervical cysts ,
- lymphangioma,
- hemangioma,
- thyroiditis,
- thyroid adenoma,
- thyroid cancer.

Therapy

- endemic goiter prevention: salt fortification with iodine
- iodine substitution
- at volume + 80ml: surgical thyroidectomy or radioablation

Complication

- the risk of oppression of surrounding structures : dyspnoea, dysphagia, upper vena cava syndrome

Links

Related articles in czech

- Onemocnění štítné žlázy: Hypotyreóza • Hypertyreóza
- Vyšetření u chorob štítné žlázy • Vyšetření funkce štítné žlázy
- Symptomatické duševní poruchy při endokrinopatiích

References

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3. LEBL, J, J JANDA and P POHUNEK, et al. *Clinical pediatrics*. 1st edition. Galén, 2012. 698 pp. 185-188. ISBN 978-80-7262-772-1 .
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